A DISSERTATION

ON

A STUDY ON ASYMPTOMATIC BACTERIURIA IN WOMEN WITH DIABETES MELLITUS

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CERTIFICATE

This is to certify that the dissertation entitled **"A study on Asymptomatic Bacteriuria in Women with Diabetes Mellitus"** is a bonafide work done by **Dr. V.PARAMASIVAM** in **M.D BRANCH I GENERAL MEDICINE** at Government Mohan Kumaramangalam Medical College, Salem, to be submitted to The Tamil Nadu Dr.M.G.R Medical University, in fulfilment of the University Rules and Regulation for the award of M.D. Degree Branch I General Medicine, under my supervision and guidance, during the academic period from January 2007 to July 2008.

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DECLARATION

I solemnly declare that this dissertation **"A study on Asymptomatic Bacteriuria in Women with Diabetes Mellitus"** was prepared by me at Government Mohan Kumaramangalam Medical College and Hospital, Salem under the guidance and supervision of **Prof. Dr.K.SATHYAMOORTHY, M.D.,** Professor of General Medicine, Govt. Mohan Kumaramangalam Medical College and Hospital Salem.

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INTRODUCTION

Diabetes mellitus comprises a group of common metabolic disorders that share the phenotype of hyperglycemia. The metabolic dysregulation associated with diabetes melliltus causes secondary pathophysiologic changes in multiple organ systems that impose a tremendous burden on the individual with diabetes and on the health care system. With an increasing incidence worldwide, diabetes mellitus will likely continue to be a leading cause of morbidity and mortality.

The complications of diabetes mellitus affect many organ systems and are responsible for the majority of morbidity and mortality associated with the disease. Diabetes and its complications produce a wide range of symptoms and signs; those secondary to acute hyperglycemia may occur at any stage of the disease, whereas those related to chronic complications begin to appear 'during the second decade of hyperglycemia. Individuals with previously undetected diabetes mellitus may present with chronic complications of diabetes mellitus at the time of diagnosis.

Individuals with diabetes mellitus exhibit a greater frequency and severity of infection. Though the introduction of insulin in the treatment of diabetes mellitus has decreased the mortality in diabetes due to infections to a great extent, the morbidity due to infections still remains high. Poorly controlled diabetics are particularly susceptible to infections of urinary tract, respiratory tract, soft tissues and skin.

Urinary tract infection is classically assumed to be a clinically relevant problem for patients with diabetes mellitus. Severe complications of urinary tract infection occur in' patients with diabetes. Some individuals with diabetes may present with a distressing picture showing definite progression of pyelonephritis characterized by evidence of systemic infection, local extension of the infection (e.g. renal and perinephric abscesses), septicemia, and severe impairment of metabolic control, which may become too difficult to manage. For these reasons, diabetes has long been considered to be a predisposing factor for urinary tract infection.

Urinary tract infections usually present with dysuria, urgency, frequency and suprapubic pain. Many urinary tract infections are

asymptomatic especially in women. Most of the symptomatic urinary tract infections are preceded by asymptomatic bacteriuria.

Asymptomatic bacteriuria is common in neonates, preschool children, pregnant women, elderly people and diabetics. Asymptomatic bacteriuria occurring in diabetes mellitus can cause serious complications like emphysematous pyelonephritis, renal papillary necrosis and renal abscess. Severe impairment of metabolic control of diabetes mellitus increases the risk of acquiring asymptomatic bacteriuria.

Various studies have studied the risk factors for asymptomatic bacteriuria in diabetic patients which include age, duration of diabetes, glycemic status and other complications of diabetes. Numerous studies have evaluated the frequency of symptomatic bacteriuria in men and women with diabetes. The prevalence of asymptomatic bacteriuria is increased in diabetics with poor metabolic control. Many investigators have recommended screening patients with diabetes to detect and then treat asymptomatic bacteriuria because of the increased frequency and severity of upper urinary tract infections in such patients.

REVIEW OF LITERATURE

Diabetes mellitus is defined as a disturbance of intermediary metabolism manifesting as chronic sustained hyperglycemia primarily due to either an absolute or a relative lack of insulin. The world today is witnessing an epidemic of diabetes mellitus. Globally and nationally, diabetes mellitus with its complications has become the most important contemporary and challenging health problem. Diabetes mellitus is the leading cause of end stage renal disease, a major cause of non-traumatic amputations, responsible for a large percentage of preventable blindness and a leading cause of cardiovascular mortality. The worldwide prevalence of diabetes mellitus has risen dramatically over the past few decades. It is projected that the number of individuals with diabetes mellitus will continue to increase in the near future.

The management of diabetes mellitus aims to achieve a good control of several metabolic parameters and a euglycemic state to avoid acute and chronic complications of diabetes mellitus. These include diabetic ketoacidosis, hyperglycemic hyperosmolar state,

neuropathy, nephropathy, retinopathy, cerebral vascular disease, coronary artery disease, peripheral vascular disease and infections. Despite the introduction of insulin, diabetic patients still have a considerably reduced life expectancy. This excess mortality is mainly due to long term complications. Good metabolic control can definitely help to delay the onset of complications as well as slow down their progression to a large extent. However, it has not been possible to completely eliminate these complications.

Individuals with diabetes mellitus exhibit a greater frequency and severity of infection. Many common infections are more frequent and severe in the diabetic population whereas several rare infections are seen almost exclusively in the diabetic population. The mortality in diabetes due to infections has been reduced to a great extent after the advent of insulin and various antibiotics. However, the morbidity due to infections still remains high. Poorly controlled diabetics are particularly susceptible to infections of urinary tract, respiratory tract, soft tissues and skin. Trivial infections may progress rapidly due to lowered immunological resistance. There are various factors which influence resistance to infections. Insulin is needed for normal metabolism of glucose to provide energy for phagocytosis and to

destroy microorganisms. Hence the consequences of insulin lack are defective diapedesis, defective leukocyte adherence, defective phagocytosis, defective enzyme activity and defective bactericidal activity. Complications of diabetes can predispose to infections. Microangiopathy results in impaired tissue perfusion which delays healing. The antibiotics fail to reach the site of infection. Peripheral neuropathy predisposes to foot trauma and secondary infection supervenes. These infected ulcers fail to heal due to associated poor peripheral circulation. Autonomic neuropathy results in bladder dysfunction and stasis which predisposes to urinary tract infections. Glycosuria and repeated catheterization are additional risk factors for urinary tract infections.

MICROBIOLOGY

Urinary tract infection, from a microbiologic perspective exists when pathogenic microorganisms are detected in the urine, urethra, bladder, kidney or prostate. In most instances, growth of more than 10^5 organisms per milliliter from a properly collected midstream clean catch urine sample indicates infection. In symptomatic patients, a small number of bacteria like 10^2 to 10^4 per milliliter may signify infection. In urine specimens collected by suprapubic aspiration or in-

and-out catheterization and in samples from a patient with an indwelling catheter, colony counts of 10^2 to 10^4 per milliliter generally indicate infection.

Acute community acquired infections are very common and occur in schoolgirls and then increase markedly in incidence with the onset of sexual activity in adolescence. The vast majority of symptomatic infections involve young women. Acute symptomatic infections are rare in men under the age of 50. The development of asymptomatic bacteriuria parallels that of symptomatic infection and is rare among men under 50 but common in women between 20 and 50. Asymptomatic bacteriuria is common among elderly men and women.

Many different microorganisms can infect the urinary tract, but by far the most common agents are the gram-negative bacilli. Escherichia coli is the most common organism. Other gram-negative rods especially Proteus, Klebsiella and Enterobacter are responsible for a smaller proportion of urinary tract infection. Serratia and Pseudomonas assume increasing importance in recurrent infections and in infections associated with urologic manipulation, calculi, or

obstruction. They play a major role in nosocomial, catheter associated infections. Proteus by virtue of urease production, and Klebsiella species through the production of extracellular slime and polysaccharides predispose to stone formation and are isolated more frequently from patients with calculi.

Gram-positive cocci play a lesser role in Urinary tract infections. However, Staphylococcus saprophyticus accounts for 10 to 15% of acute symptomatic urinary tract infections in females. Enterococci occasionally cause actue uncomplicated cystitis in women. More commonly, enterococci and Staphylococcus aureus cause infections in patients with renal stones or previous instrumentation or surgery. Isolation of S. aureus from the urine should arouse the suspicion of bacteremic infection of the kidney. Sexually transmitted urethritis-producing agents such as Chlamydia trachomatis, Neisseria gonorrhoeae, and Herpes simplex virus are etiologically important in young, sexually active women with new sexual partners.

The causative role of nonbacterial pathogens in urinary tract infections remains poorly defined. Ureaplasma urealyticum has

frequently been isolated from the urethra and urine of patients with acute dysuria. Colonization of the urine of catheterized or diabetic patients by candida and other fungal species is common and sometimes progresses to symptomatic invasive infection.

PATHOGENESIS AND SOURCES OF INFECTION

In the vast majority of urinary tract infections, bacteria gain access to the bladder via the urethra. Ascent of bacteria from the bladder may follow and is probably the pathway for most renal parenchymal infections. The vaginal introitus and distal urethra arc normally colonized by diphtheroids, streptococcal species, lactobacilli and staphylococcal species but not by the enteric gram negative bacilli that commonly cause urinary tract infections. In females prone to the development of cystitis, however, enteric gram negative bacilli residing in the bowel colonize the introitus, the periurethral skin, and the distal urethra before and during episodes of bacteriuria. The factors that predispose to periurethral colonization with gram negative bacilli remain poorly understood, but alteration of the normal vaginal flora by antibiotics, other genital infections or contraceptives especially spermicides appears to play an important role. Loss of the normally dominant H_2O_2 producing lactobacilli in the vaginal flora

appears to facilitate colonization by E. coli. Small number of periurethral bacteria probably gain entry to the bladder frequently, a process that is facilitated in some cases by urethral massage during intercourse. Whether bladder infection ensues depends on interacting effects of the pathogenicity of the strain, the inoculum size and the local and systemic host defense mechanisms.

Under normal circumstances, bacteria placed in the bladder are rapidly cleared, partly through the flushing and dilutional effects of voiding but also as a result of the antibacterial properties of urine and the bladder mucosa. Owing mostly to a high urea concentration and high osmolarity, the bladder urine of many normal persons inhibits or kills bacteria. Polymorphonuclear leukocytes enter the bladder epithelium and the urine soon after infection arises and play a role in clearing bacteriuria. Hematogenous pyelonephritis occurs most commonly in debilitated patients who are either chronically ill or receiving immunosuppressive therapy. Metastatic staphylococcal or candidal infections of the kidney may follow bacteremia or fungemia, spreading from distant foci of infection in the bone, skin, vasculature or elsewhere.

CONDITIONS AFFECTING PATHOGENESIS

GENDER AND SEXUAL ACTIVITY

The female urethra appears to be particularly prone to colonization with colonic gram-negative bacilli because of its proximity to the anus, its short length and its termination beneath the labia. Sexual intercourse causes the introduction of bacteria into bladder and is temporally associated with the onset of cystitis; it thus appears to be important in the pathogenesis of urinary tract infections in younger women. Use of spemricide-coated condoms alters the normal introital bacterial flora and has been associated with marked increases in vaginal colonization with E.coli and in the risk of urinary tract infection.

PREGNANCY

Urinary tract infections are detected in 2 to 8% of pregnant women[10]. Symptomatic upper tract infections are unusually common during pregnancy. Pregnant women with asymptomatic bacteriuria subsequently develop pyelonephritis. This predisposition to upper tract infection during pregnancy results from decreased urethral tone, decreased ureteral peristalsis, and temporary incompetence of the vesicoureteral valves.

OBSTRUCTION

Any obstruction to the free flow of urine like tumour, stricture, or stone results in hydronephrosis and a greatly increased frequency of urinary tract infection. Infection superimposed on urinary tract obstruction may lead to rapid destruction of renal tissue.

NEUROGENIC BLADDER DYSFUNCTION

Interference with the nerve supply to the bladder as in spinal cord injury, diabetes mellitus, tabes dorsalis and other diseases may be associated with urinary tract infection. The infection is favoured by the prolonged stasis of urine in the bladder[19].

VESICOURETERAL REFLUX

Vesicoureteral reflux which is defined as reflux of urine from the bladder cavity up into the ureters and sometimes into the renal pelvis occurs during voiding or with elevation of pressure in the bladder. An anatomically impaired vesicoureteral junction facilitates reflux of bacteria and thus upper tract infection.

BACTERIAL VIRULENCE FACTORS

Bacterial virulence factors markedly influence the likelihood that a given strain, once introduced into the bladder will cause urinary tract infection. Most E.coli strains that cause urinary tract infections belong to a small number of specific O, K, and H serogroups. These uropathogenic clones have accumulated a number of virulence genes that are often closely linked on the bacterial chromosome in virulence islands. Adherence of bacteria to uroepithelial cells is a critical first step in the initiation of infection. For both E.coli and Proteus, fimbriae mediate the attachment of bacteria to specific receptors on epithelial cells. The attachment of bacteria to uroepithelial cells initiates a number of important events in the mucosal epithelial cells including secretion of interleukin 6 and interleukin 8 and induction of both apoptosis and epithelial cell desquamation. Besides fimbriae, uropathogenic E.coli strains usually produce haemolysin and aerobactin, which are resistant to the bactericidal action of human serum. Nearly all E.coli strains causing acute pyelonephritis and most of those causing acute cystitis arc uropathogenic.

GENETIC FACTORS

Host genetic factors influence the susceptibility to urinary tract infection. The number and type of receptors on uroepithelial cells to which bacteria may attach are at least in part genetically determined. Nonsecretors of blood group antigens are at increased risk of recurrent urinary tract infection. This predisposition may relate to a different profile of genetically determined glycolipids on uroeithelial cells.

SERIOUS COMPLICATIONS

Diabetic patients with asymptomatic bacteriuria are at high risk of developing serious complications of urinary infection such as emphysematous cystitis, pyelonephritis, papillary necrosis or renal and perinephric abscess. There is a four fold increased risk for the development of pyelonephritis and a two fold increased risk for the development of renal corticomedullary abscess among diabetic patients.

PAPILLARY NECROSIS

When infection of the renal pyramids develops in association with vascular diseases of the kidney or with urinary tract obstruction, renal papillary necrosis is likely to result. Patients with diabetes seem

peculiarly susceptible to this complication. Hematuria, flank pain, chills and fever are the most common presenting symptoms. Renal papillary necrosis is often bilateral. Acute renal failure with oliguria or anuria sometimes develops. Rarely, sloughing of a pyramid may take place without symptoms in a patient with chronic Urinary tract infection, and the diagnosis is made when the necrotic tissue is passed in the urine or identified as a ring shadow on pyelography. If renal function deteriorates suddenly in a diabetic individual, the diagnosis of renal papillary necrosis should be suspected even in the absence of fever or pain.

EMPIIYSEMATOUS PYELONEPHRITIS

Emphysematous pyelonephritis is an unusual complication that almost always occur in diabetic patients. Emphysematous pyelonephritis is usually characterized by a rapidly progressive clinical course, with high fever, leukocytosis, renal parenchymal necrosis, and accumulation of fermentative gases in the kidney and perinephric tissues. Most patients have pyuria and glycosuria. Escherichia coli causes most cases, but occasionally Enterobacteriaceae are isolated. Gas in tissues can often be seen on plain films and can be confirmed by computed tomography. Surgical

resection of the involved tissue in addition to systemic antimicrobial therapy is usually needed to prevent mortality in emphysematous pyelonephritis.

EMPHYSEMATOUS CYSTITIS

Emphysematous cystitis occurs primarily in diabetic patients, usually in association with Escherichia coli or facultative Gram-negative rods and often in relation to bladder neck obstruction. Patients with this condition are less severely ill and have less rapidly progressive disease than those with emphysematous pyelonephritis. The patient typically reports abdominal pain, dysuria, frequency and in some pneumaturia. Computed tomography shows gas within both the bladder lumen and bladder wall. Conservative therapy with systemic antimicrobial agents and relief of outlet obstruction are effective.

PERINEPHRIC AND RENAL ABSCESS

Perinephric and renal abscesses are quite uncommon[26]. Before the advent of antibiotics, most renal and perirenal abscesses were hematogenous in origin, with Staphylococcus aureus most commonly recovered. Nowadays, > 75% of perinephric and renal abscesses arise

from an initial urinary tract infection. Infection ascends from the bladder to the kidney, with pyelonephritis occurring first. Bacteria may directly invade the renal parenchyma from medulla to cortex. Local vascular channels within the kidney may also facilitate the transport of organisms. Areas of abscess developing within the parenchyma may rupture into the perinephric space. Many patients with perinephric abscess have concomitant nephrolithiasis producing local obstruction to urinary flow. The organisms encountered in perinephric and renal abscesses are Escherichia coli, Proteus species and Klebsiella species. Escherichia coli have unique virulence properties in the urinary tract which promote adherence to uroepithelial cells. The urease of Proteus species splits urea thereby creating a more alkaline and hospitable environment for bacterial proliferation. The presentation of perinephric and renal abscesses is quite nonspecific. Flank pain and abdominal pain are common. Fever is usually present. The diagnosis is usually made by renal ultrasonography or abdominal computed tomography.

TREATMENT PRINCIPLES

The following principles[25] underlie the treatment of urinary tract infections:

1. Except in uncomplicated cystitis in women, a quantitative urine culture or a Gram stain should be performed to confirm infection before treatment is begun. When culture results become available, antimicrobial sensitivity should be used to direct therapy.
2. Factors predisposing to infection such obstruction and calculi should be identified and corrected if possible.
3. Relief of clinical symptoms does not always indicate bacteriologic cure.
4. Each course of treatment should be classified as a failure or a cure. Recurrent infections should be classified as same strain or different strain and as early (occurring within 2 weeks of the end of therapy) or late.
5. Uncomplicated infections confined to the lower urinary tract respond to short courses of therapy while upper tract infections require longer treatment.
6. Despite increasing resistance, community-acquired infections, especially initial infections are usually due to more antibiotic sensitive strains.

7. In patients with repeated infections, instrumentation or recent hospitalization, the presence of antibiotic-resistant strains should be suspected.

METHODS OF URINE SAMPLE COLLECTION

Urine sample can be collected by:

1. Clean catch midstream specimen
2. Suprapubic aspiration of urine
3. Bladder catheterization

Clean catch mid stream specimen is collected as follows: patient must have full bladder. Patient removes underclothing and stands legs on either side of toilet. Separate labia with left hand and clean the vulva front to back with sterile swab. Void downward into toilet until half done. Without interrupting stream catch urine in sterile bottle and complete voiding. This method is followed because the distal urethra normally comains bacteria and so the first voided urine is contaminated with the bacteria. So midstream urine collection is done.

Suprapubic aspiration of urine is done when it is not possible to obtain uncontaminated samples or in symptomatic patients with low bacterial counts. This method is not routinely followed. Patient must have a full bladder which can be percussed and if still in doubt, localize bladder using ultrasound. With patient lying supine, choose site in midline 2.5 cm above pulbic symphysis and clean skin with spirit impregnated sterile gauze. Insert a 21 gauge 1.5" needle attached to a 10 ml syringe directly downwards and aspirate urine. Withdraw needle; and collect urine. The procedure may also be done under local anaesthesia.

Urine samples can also be obtained by "in-and-out" bladder catheterization; But this method is nearly unnecessary since catheterization may also introduce infection.

Urine being an excellent medium for growth of most microorganisms, it must be plated immediately or refrigerated at 4°C.

TESTS FOR DETECTION OF URINARY TRACT INFECTION

1. Gram staining

It is least expensive and a reliable screening method for identification of microorganisms. A drop of well mixed urine is allowed to dry. The smear is Gram stained and examined under oil immersion objective. Presence of atleast one organism per oil immersion field, examining 20 fields correlated with significant bacteriuria.

2. Griess Nitrite test

This test is based on the absence of nitrite in normal urine. The presence of nitrite, detected by a simple test indicates the presence of nitrate reducing bacteria in urine. A positive test suggests the presence of atleast 10^5 organisms per ml of urine. This test detects E.coli, Klebsiella, Proteus, Staphylococcus and Pseudomonas species. False negative test occurs in the presence of some yeast, some gram positive cocci and urinary ascorbic acid.

3. Catalase test

This test depends on the generation of oxygen bubbles by catalase produced by the bacteria when hydrogen peroxide is added to

the infected urine. False positive results occur in the presence of haematuria.

4. Triphenyl tetrazolium chloride test

The respiratory activity of growing bacteria reduce 2,3,5 triphenyl tetrazolium chloride to pink red insoluble precipitate.

5. Glucose oxidase test

This test depends on the bacterial metabolism of glucose normally present in urine. In the presence of infection, glucose is not detected. False positive results occur in glycosuric patients.

6. Leucocyte esterase test

The leucocyte esterase test detects esterases released from degraded white blood cells. This is an indirect test for bacteriuria. This test is rapid and requires little technical expertise.

7. Dipslide culture methods

Agar coated slides are immersed in urine or even exposed to the stream of urine during voiding, incubated and the growth estimated by colony counting or by colour change of indicators.

8. Bac-T screen bacteriuria detection device

In this method urine is forced through a filter paper, which retains microorganisms, somatic cells and other particles. A dye is then added to the filter paper to visualize the particulate matter that has adhered. The intensity of the color relates to the number of particles. This procedure is very rapid and has been shown to detect even 10^2 organisms per ml. None of the screening methods are as reliable as urine culture.

QUANTITATIVE URINE CULTURE

Pour plate dilution technique:

This is an extremely accurate method but time consuming. It is used as a standard of comparison for other methods. Here double dilution series of urine are spread over the culture plate. The number of colonies in each plate is read in 24 hours ad 48 hours and colonies are calculated.

Surface culture methods:

Serial 10 fold dilution of urine are plated by surface culture method. Number of colonies is calculated at the end of 24 hours and 48 hours. These methods are too complicated for routine diagnosis.

Bacteriologic loop technique:

This is the most commonly employed method. In this standard platinum loops or disposable sterile loops designed to deliver either 0.01 ml or 0.001 ml of urine are used. The urine should be mixed thoroughly before plating. Flame an inoculating loop and allow it to cool without touching any surface. Insert the loop vertically into the urine to allow urine to adhere to the loop. Spread the loopful of urine to the surface of blood agar. The loop is touched to the center of the plate from which the inoculum is spread in a line across the diameter of the plate. Without flaming insert the loop vertically into the urine again for transfer of a loopflll to an indicator medium. Incubate plates for atleast 24 hours at 35° to 37° C in air. The colonies are counted on each plate. The number of colonies are multiplied by 1000 (if a 0.001 ml loop is used) or by 100 (if a 0.01 ml loop is used) to determine the number of microorganisms per milliliter in the original specimen. The former medium gives quantitative measurement of bacteriuria and the latter a presumptive diagnosis of the bacterium. The isolates are identified by their properties. Reincubate plates with no growth or scanty growth for an additional 24 hours before discarding the plates.

ANTIBIOTIC SENSITIVITY TESTS

Pathogenic bacteria exhibit very great variation in susceptibllity to antibiotics and chemotherapeutic agents. Therefore, it is essential to determine the susceptibility of isolates of pathogenic bacteria to antibiotics that are most likely to be used in treatment. Antibiotic sensitivity is routinely done by Kirby-Bauer disc diffusion method[15]. The disc diffusion method uses filter paper discs 6.00 mm in diameter, charged with appropriate concentrations of the drugs. A suitable dilution of a broth culture or a broth suspension of the test bacterium is flooded on the surface of a solid medium (Mueller-Hinton agar). The plate is tilted to ensure uniform spreading and the excess broth is pipetted off. After drying the plate, antibiotic discs are applied with sterile forceps. After overnight incubation, the degree of sensitivity is determined by measuring the zones of inhibition of growth around the discs. Growth will be inhibited around discs containing antibiotics to which the bacterium is susceptible but not around those to which it is resistant. The results are reported as 'sensitive' or 'resistant' to the different drugs.

REVIEW OF ARTICLES

1. Asymptomatic bacteriuria among outpatients with diabetes mellitus in an urban black population. Cent Afr J Med. 2002 .Jul-Aug, 48(7-8):78-82.

The objective was to determine the prevalence of asymptomatic bacteriuria in individuals afflicted by diabetes mellitus, the antibiotic susceptibility of the microbial isolates and the association of host factors with asymptomatic bacteriuria. This was a prospective cross sectional study. There were 176 participants. The prevalence of asymptomatic bacteriuria was 32% in the diabetics and 11% in nondiabetic participants. The commonest bacterial organism isolated in participants afflicted by diabetes mellitus was Escherichia coli (26%) followed by Staphylococcus aureus (21%), Streptococcus group B (14%), Streptococcus group D and non lactose fermenting coliforms (7% respectively). Other isolates were micrococccus and Pseudomonas (5% respectively), Klebsielia and Proteus (2% respectively). Gentamicin, nitrofurantoin, ampicillin were the most effective antimicrobials in the majority of the isolates. Certain isolates exhibited some bacterial resistance to conventional antibiotics. An

association was found between bacteriuria and glucosuria and between leucocyturia and bacteriuria. The study concludes that the prevalence of asymptomatic bacteriuria is increased in diabetes.

2. Asymptomatic bacteriuria in type 2 diabetics women. Rev Med Chil. 2002 Sep; 130 (9): 1001-7

The aim of the study was to determine the frequency of asymptomatic bacteriuria in type 2 diabetic women. Fifty women with type 2 diabetes and 50 nondiabetic women were studied. In aseptic conditions, morning midstream urine specimens were obtained for microbiological analysis. There was microbial growth in 40% of samples from diabetics and 6% of samples from controls. Asymptomatic bacteriuria was present in 32% of diabetics and 4% of controls. E.coli was the most commonly isolated strain. Klebsiella, coagulase negative Staphylococcus and Enterococcus were also isolated. Women with positive cultures had a longer lasting diabetes than those with negative cultures. There was no association between urine microbiological results and glycosylated hacmoglobin, fasting blood glucose and chronic complications. The study concludes that asymptomatic bacteriuria is highly prevalent among diabetic women.

3. Consequences of asymptomatic bacteriuria in women with diabetes mellitus. Arch Intern Med. 2001:161:1421-1427.

The objective was to compare women with diabetes mellitus with and without asymptomatic bacteriuria for the development of symptomatic urinary tract infection, renal function and secondary complications of diabetes mellitus. 636 diabetic women were studied. The prevalence of asymptomatic bacteriuria at baseline was 26%. Follow up results were available for 589 of the 636 women. Of these, 20% developed a symptomatic urinary tract infection. Women with type 2 diabetes and asymptomatic bacteriuria at baseline had an increased risk of developing a urinary tract infection during the 18 month follow up period.

4. Pathogenesis of bacteriuria in women with diabetes mellitus. Int J Antimicrob Agents. 2002 Jun: 19(6): 539~45.

Women with diabetes mellitus have asymptomatic bacteriuria and symptomatic urinary tract infection more often than women without diabetes mellitus. The increased prevalence of bacteriuria in diabetic patients can be the result of differences in the host responses between diabetic and nondiabetic patients, or a difference in the infecting bacterium itself. The study shows that the increased

prevalence of asymptomatic bacteriuria in diabetic women is not the result or a difference in bacteria, because the same number of virulence factors was found in the infecting Escilerichia coli in diabetic women with asymptomatic bacteriuria, as listed in the literature for nondiabetic patients with asymptomatic bacteriuria. The study found that bacterial growth in vitro was increased after the addition of different glucose concentrations, as found in urine of poorly controlled patients. The study also demonstrated that women with both diabetes mellitus and asymptomatic bacteriuria have a lower urinary cytokine and leucocyte concentrations than women without diabetes mellitus but with asymptomatic bacteriuria. The study also found that E.coli expressing type 1 fimbriae adhere better to urocpithe1ial cells of women with diabetes mellitus compared with the cells of women without diabetes mellitus.

5. Asymptomatic bacteriuria in women with diabetes: influence of metabolic control. Clinical Infectious Diseases 2004;38:c4I-c45.

The study was conducted during the period of January 1997 to December 2000 at Pisa General hospital, Italy. 228 women with diabetes were screened for bacteriuria. A control group of 146 women without diabetes was also evaluated. The frequency of significant

bacteriuria was 17.5% among diabetic women. The presence of higher glycated haemoglobin levels was a significant risk factor for significant bacteriuria in women with diabetes. Severe impairment of metabolic control of type 2 diabetes increases the risk of acquiring asymptomatic bacteriuria.

6. Asymptomatic bacteriuria in patients with diabetes - enemy or innocent visitor? NEJM Volume 347:1617-1618.

Eighty percent of women with diabetes and bacteriuria have been shown to have renal parenchymal infection by seven weeks after initial testing. A variety or factors may contribute to the increased frequency of urinary tract infection in diabetic women. Bladder dysfunction as a result of diabetic neuropathy and cystopathy are the most important.

7. Asymptomatic bacteriuria and hemoglobin AL. JK Schmitt et all. Diabetes Care, Vol 9, Issue 5518-520.

The authors measured hemoglobin A I and performed clean-catch urine cultures in 752 patients (411 men and 341 women) with non-insulin dependent diabetes mellitus attending an outpatient diabetes clinic. Prevalence of bacteriuria was significantly greater in diabetic women than in controls (9.1 vs 5.0%, P <0.001) but not in

diabetic men. Risk of bacteriuria was not related to level of HbAL at the time of urine culture. However, mean duration of diabetes mellitus was significantly greater in diabetic women with bacteriuria than in those without infection and the prevalence of bacteriuria was significantly greater in patients with complications of long standing diabetes mellitus than in those without complications.

8. Adherence of Type-1 fimbriated Escherichia coli to uroepithelial cells. More in diabetic women than in control subjects. Suzanne E. Geerlings et al. Diabetes care 25 : 1405 – 1409, 2002.

It was hypothesized that E.coli adhere more to the uroepithelial cells of diabetic women, either because of substances excreted in the urine (e.g. albumin, glucose and Tamm Horsfall protein) or because of a difference in the uroepithelial cells. A T24 bladder cell line and uroepithelial cells of 25 diabetic women and 19 control subjects were incubated with 3 different E.coli strains. They were Type-l fimbriated E.coli, P-fimbriated E.coli and nonfimbriated E.coli. Type-I fimbriated E.coli adhere more to diabetic than to control uroepithelial cells.

9. Prevalence of asymptomatic bacteriuria in subjects with NIDDM in San Luis valley of Colorado. EM Keane et a!. Diabetes Care, Vol 11, Issue 9 708-712.

This study examined whether non insulin dependent diabetic subjects have an increased prevalence of asymptomatic bacteriuria compared with subjects with normal glucose tolerance. Presence of asymptomatic bacteriuria was determined by testing the subjects urine with a reagent strip test for nitrite and leucocyte esterase. Among diabetic subjects, prevalence of bacteriuria increased with longer disease duration but was not affected by measures of glucose control. The study concludes that NIDDM increases the prevalence of bacterial colonization of the urine and, therefore, probably also increases the risk of symptomatic urinary tract infection.

10. Urinary tract infections in adults with diabetes. Ronald A et al. Int J Antimicrob Agents. 2001 Apr; 17(4):287-92.

Urinary tract infection is a major disease burden for many patients with diabetes. Asymptomatic bacteriuria is several fold more common among women and acute pyelonephritis is five to ten times more common in both sexes. The complications of pyelonephritis are

also more common in patients with diabetes. These complications include acute papillary necrosis, emphysematous pyelonephritis and bacteremia with metastatic localization to other sites.

AIMS AND OBJECTIVES

1. To estimate the prevalence of asymptomatic bacteriuria among women with diabetes mellitus.
2. To identify the causative organisms responsible for asymptomatic bacteriuria in women with diabetes mellitus.
3. To determine the antibiotic susceptibility of the isolated organisms.
4. To verify whether there is a correlation between bacteriuria and the known duration of diabetes.
5. To verify whether there is a correlation between bacteriuria and the metabolic control of diabetes.

MATERIALS AND METHODS

SETTING

The study was conducted in the medical wards of Government Mohan Kumaramangalam College Hospital, Salem.

COLLABORATING DEPARTMENTS

The study was conducted with collaboration from the Department of Diabetology and the Department of Microbiology of Government Mohan Kumaramangalam College Hospital, Salem.

DESIGN OF STUDY

Prospective cross sectional study,

PERIOD OF STUDY

The study was conducted on patients between January 2007 and June 2008.

SAMPLE SIZE

The study was conducted on 200 diabetic women and 75 age matched nondiabetic women.

SELECTION OF STUDY SUBJECTS AND CONTROL

Two hundred women with diabetes mellitus were enrolled in the study. These women were patients at the Department of Internal Medicine, Government Mohan Kumaramangalam College Hospital, Salem. A randomly selected control group of 75 healthy women without diabetes was also evaluated in the same period.

EXCLUSION CRITERIA

- Patients with symptoms of urinary tract infection.
- Bladder catheterization during the 2 months before enrollment in the study.
- Instrumentation of urogenital tract during the 2 months before enrollment in the study.
- Pregnancy.
- Use of antimicrobial drugs during the previous 14 days.
- Recent hospitalization or surgery within the past 4 months.
- Gynecological infections.

DETAILS OF STUDY SUBJECTS

During initial visit relevant history was elicited from patients regarding age, known duration of diabetes, medication, pregnancy,

history for urinary tract infection, history of previous catheterization, instrumentation, history of white discharge and history of pruritus vulva. Gynecological examination was carried out to rule out infections of the reproductive tract.

During subsequent visit, fasting and post prandial plasma glucose tests were done on the patients and control group. Clean catch mid stream urine specimen was collected and culture and microscopic tests were done. The urine samples were collected during the non-menstrual period.

DEFINITIONS

Diabetes mellitus: Diagnosis of diabetes mellitus was made in accordance with the criteria of the: American Diabetes Association. A patient was diagnosed to be diabetic if she had (a). Symptoms of diabetes plus random plasma glucose > 200 mg/dl or (b). Fasting plasma glucose > 126 mg/dl or (c) Two-hour plasma glucose > 200 mg/dl or (d) the use or glucose lowering medications (oral agents or insulin).

Asymptomatic bacteriuria: Asymptomatic bacteriuria is defined as the presence of at least 10^5 colony forming units per ml of 1 or 2 bacterial species in a culture of clean-voided midstream urine specimen from an individual without symptoms of urinary tract infection.

Contaminated Urine: Contaminated urine is defined as the presence of at least 3 different microorganisms in 1 urine specimen.

Leucocyturia is defined as the presence of more than 10 leucocytes per high power field in the sediment of centrifuged urine.

DETAILS OF MATERIALS

Urine specimen was collected by clean catch mid stream method. Patients were explained about the methods of collecting clean catch midstream urine and elderly female patients were provided with a nursing assistant for cleaning the external genitalia. Urine was collected in a sterile wide mouthed screw cap bottle for culture purpose and another sample collected for microscopic examination of leucocyturia.

The number of white blood cells per cubic millimeter of urine was estimated using haemocytometer in microscope.

Urine samples were streaked on nutrient agar and MacConkey agar for confirmation of bacterial growth. Identification of urine isolates was performed using conventional methods and the invitro susceptibility to antimicrobial drugs was tested by the Kirby-Bauer disc diffusion method. Antibiotic sensitivity tests were done using the standard amounts of antibiotics (Ampicillin, Gentamicin; Cotrimoxazole, Ciprofloxacin, Cefotaxime, Doxycycline, Norfloxacin, Ceftriaxone, Amikacin and Nalidixic acid) and report was obtained at the end of 48 hours.

Fasting and 2 hour postprandial venous blood samples were withdrawn and plasma glucose values were estimated.

ETHICAL APPROVAL

Ethical approval was obtained from the Ethical committee of Government Mohan Kumaramangalam College Hospital headed by the Dean, Government Mohan Kumaramangalam College Hospital, Salem.

CONSENT

Informed consent was obtained from the patients and the control group.

LIMITATIONS

Glycated haemoglobin was not estimated due to technical difficulties.

STATISTICAL ANALYSIS

Statistical analysis was done using standard statistical packages.

RESULTS

In this study, 200 women with diabetes mellitus and 75 age matched non-diabetic women (control group) were studied.

The age distribution of cases and controls is shown in Table 1.

Table 1. Age-distribution of cases and controls

Age group (Yrs.)	Cases	Controls
21-30	3	1
31-40	29	12
41-50	63	23
51-60	53	17
61-70	38	16
>70	14	6
TOTAL (n)	**200**	**75**

The age of the patients was 52.1 ± 11.1 years. The youngest in the study group was 21 years and the oldest in the study group was 75 years. The age of the control group was 51.7 ± 11.9 years. The youngest in the control group was 26 years and the oldest in the control group was 75 years.

DURATION OF DISEASE

The known duration of diabetes mellitus in the study group is shown in Table 2.

Table 2. Duration of disease among diabetic women

Duration of diabetes (yrs)	No. of cases
0-5	69
6-10	66
11-15	40
16-20	17
> 20	8
TOTAL (n)	**200**

The duration of the disease among women with diabetes mellitus was 7.8 ± 6.0 years. More than 50% of the study group had a duration of diabetes of less than 10 years.

GLYCEMIC CONTROL

For the entire study group (n=200) comprising of diabetic women, both fasting and postprandial plasma glucose were estimated. The values are tabulated in Table 3 and Table 4.

Table 3. Fasting plasma glucose values in study group

Fasting plasma glucose (mg/dl)	No. of Patients
< 80	24
81-120	78
121-160	50
161-200	32
201-240	12
> 240	4
TOTAL	**200**

Table 4. Postprandial plasma glucose values in study group

Postprandial plasma glucose	No. of Patients
120-160	36
161-200	63
201-240	50
241-280	26
281-320	19
> 320	6
TOTAL	**200**

The fasting plasma glucose was 126.9 ± 44.0 mg/dl (Range 70-260 mg/dl) and the postprandial plasma glucose was 207.7 ± 54.9 mg/dl (Range 124-360 mg/dl).

Table 5 summarises the important demographic and clinical features of women with diabetes in this study.

Table 5. Demographic and clinical features of study group

n	**200**
Age (Yrs)	52.1 ± 11.1
Duration (Yrs)	7.8 ± 6.0
Height (m)	1.5 ± 0.3
Weight (Kg)	56.1 ± 7.8
BMI (Kg / m^2)	24.7 ± 4.6
Fasting plasma glucose (mg / dl)	126.9 ± 44.1
Postprandial plasma glucose (mg / dl)	207.7 ± 54.9

BACTERIURIA

The rate of significant bacteriuria was 34% (68 out of 200) among diabetic women and 8% (6 out of 75) among those in control group ($P < 0.05$).

Diabetic women evaluated for bacteriuria	=	200
Positive urine culture (ASB +)	=	68
Negative urine culture (ASB-)	=	132
Non-diabetic women (control group) evaluated		
For bacteriuria	=	75
Positive urine culture (ASB+)	=	6
Negative urine culture (ASB -)	=	69

The prevalence of baceriuria among diabetic and non-diabetic women is shown in Fig. 1 and Fig. 2.

LEUCOCYTURIA

Of the total study group (n=200), 68 (34%) diabetic women were ASB+. In 82% of positive urine culture (56 out of 68), leucocyturia was present. In the control group, leucocyturia was present in 83% (5 out of 6). The prevalence of leucocyturia among bacteriuric women is shown in Fig. 3 and Fig. 4.

Age distribution of bacteriuric and non-bacteriuric diabetic women

The age distribution of bacteriuric and non-bacteriuric women is tabulated in Table 6.

Table 6. Age-distribution of bacteriuric and non-bacteriuric Diabetic Women

Age group (Yrs.)	ASB+	ASB-
21-30	0	3
31-40	8	21
41-50	20	43
51-60	16	37
61-70	15	23
> 70	9	5
TOTAL (n)	**68**	**132**

The age of bacteriuric and non- bacteriuric diabetic women was 53.8 ± 12.0 years and 51.2 ± 10.6 years respectively ($P < 0.05$). As age advances, there is an increased risk of asymptomatic bacteriuria among diabetic women shown in Fig. 5.

Duration of diabetes among bacteriuric and non-bacteriuric women

The duration of diabetes among bacteriuric and non-bacteriuric women in the study group is tabulated in Table 7.

Table 7. Duration of diabetes among bacteriuric and non-bacteriuric Women

Duration of disease (Yrs.)	ASB+	ASB-
0-5	19	50
6-10	20	46
11-15	17	23
16-20	8	9
> 20	4	4
TOTAL (n)	**68**	**132**

The duration of diabetes among bacteriuric and non-bacteriuric women was 9.1 ± 6.4 years and 7.1 ± 5.7 years respectively ($P < 0.05$). Asymptomatic bacteriuria was more prevalent among diabetic women with longer duration of diabetes as shown in Fig. 6.

GLYCEMIC CONTROL

The fasting plasma sugar among bacteriuric women was 156.9 ± 45.0 mg/dl and among non-bacteriuric women was 111.4 ± 34.7 mg/dl ($P < 0.05$).

The postprandial plasma sugar among bacteriuric women was 242.8 ± 53.1 mg/dl and among non-bacteriuric women was 192.1 ± 46.6 mg/dl ($P < 0.05$).

Asymptomatic bacteriuria was more prevalent among diabetic women with poor glycemic control as shown in Fig. 7 & Fig. 8.

Table 8 summarises the characteristics of bacteriuric and non-bacteriuric women.

Table 8. Demographic and clinical characteristics of diabetic women with and without bacteriuria

Characteristic	ASB +	ASB -	P Value
n	68	132	
Age (years)	53.8 ± 12.0	51.2 ± 10.6	< 0.05
Duration of diabetes	9.1 ± 6.4	7.1 ± 5.7	< 0.05
Fasting plasma glucose (mg/dl)	156.9 ± 45.0	111.5 ± 34.7	< 0.05
Postprandial plasma glucose (mg/dl)	242.8 ± 53.1	192.1 ± 46.6	< 0.05

MICROBIAL ISOLATES

Escherichia coli was the causative agent of asymptomatic bacteriuria in 56 (82%) of 68 women with diabetes mellitus (Fig. 9). Klebsiella pneumoniae was the causative in 6 patients, Coagulase negative Staphylococci in 4 patients and Proteus mirabilis in 2 patients. In the control group, Escherichia coli was the causative in 5 women and Coagulase negative Staphylococci in 1 woman. The microbial pattern in women with bacteriuria in both study and control group is shown in Table 9.

Table 9. Microbial pattern in women with bacteriuria

Microorganism	Cases	Controls
Escherichia coli	56	5
Klebsiella pneumoniae	6	0
Coagulase negative Staphylococci	4	1
Proteus mirabilis	2	0
TOTAL (n)	**68**	**6**

ANTIMICROBIAL SUSCEPTIBILITY

Majority of the isolates recovered from diabetic women were susceptible to conventional antimicrobial drugs. Certain isolates showed resistance to antimicrobial drugs.

35% of the isolates were resistant to Ampicillin. 23% of the isolates were resistant to Ciprofloxacin. 11% of the isolates were resistant to Norfloxacin. 16% of the isolates were resistant to Nalidixic acid.

The organisms recovered from women with and from those without diabetes showed similar resistance pattern to antimicrobial drugs.

DISCUSSION

In this study, it was found that the prevalence of asymptomatic bacteriuria is higher in women with diabetes than in women without diabetes (34 vs. 8%). Early studies demonstrated no differences in the prevalence of asymptomatic bacteriuria between women with and women without diabetes, but, in the majority of recent studies, a high frequency of asymptomatic bacteriuria among women with diabetes has been reported. In the study by Geerlings et al[4], the prevalence of asymptomatic bacteriuria among diabetic women was 26% and among non-diabetic women was 6%. In a similar study by Makuyana et al[13], conducted in an urban black population, the prevalence of asymptomatic bacteriuria among diabetic women was 32% and in the control group comprising of non-diabetic women, the prevalence or asymptomatic bacteriuria was 11 %. Keane et al[11] found a 3.5 fold increased prevalence of asymptomatic bacteriuria among diabetic women in their study conducted in San Luis Valley of Colorado. Barnabas Rozsai et al[11] in their study found a prevalence of asymptomatic bacteriuria among diabetic women to be 10.1 % which

was statistically significant when compared with non-diabetic women (2.6%).

Age is a well-known risk factor for bacteriuria in women without diabetes. In the present study also, age was an important risk factor for asymptomatic bacteriuria in diabetic women. Few of the earlier studies, however, have not shown an increased incidence of asymptomatic bacteriuria in elderly women 'with diabetes. In the study by Vejlsgaard[24], there was no increased incidence of asymptomatic bacteriuria in elderly women. In the recent study by Geerlings et al[4], there was a statistically significant increased prevalence among elderly women. In the study by Barnabas Rozsai, asymptomatic bacteriuria among diabetic women tended to increase with age.

In the present study, a significant correlation was found between the known duration of diabetes mellitus and asymptomatic bacteriuria in women with diabetes. The duration of diabetes mellitus among bacteriuric and nonbacteriuric women was 9.1 ± 6.4 years and 7.1 ± 5.7 years respectively ($P < 0.05$). Geerlings et al[4] found a statistically significant correlation between the duration of diabetes mellitus and asymptomatic bacteriuria among diabetic women.

Vejlsgaard[24] and Keane[1] et al[11] found a correlation among duration of diabetes and the presence of asymptomatic bacteriuria in diabetic patients. Mean duration of diabetes mellitus was significantly greater in diabetic women with bacteriuria than in those without infection (9.9 + 1.5 vs. 5.4 ± 0.4 years, $P < 0.02$) in the study by Schmitt et al[20]. The higher incidence of asymptomatic bacteriuria in women with longer duration of diabetes mellitus may be contributed to autonomic dysfunction[19] of the bladder which promotes stasis of urine and infection.

In this study, a significant correlation was found between metabolic control of diabetes and asymptomatic bacteriuria in women with diabetes. Both fasting and postprandial plasma glucose values correlated with presence of bacteriuria. This means that significant impairment of metabolic control of the disease increases the risk of developing asymptomatic bacteriuria. Hoepelman et al found that bacterial growth in vitro was increased after the addition of different glucose concentrations as found in urine of poorly controlled patients. Mendoza et al[14] did not find any association between bacteriuria and fasting blood glucose values. In the study by Geerlings et al, the

glycated haemoglobin levels did not influence the risk of asymptomatic bacteriuria. Schmitt et al[20] did not find any relation between glycated hemoglobin and risk of bacteriuria. In a study of 110 patients, Kelestimur et al[12] found a significant association between bacteriuria and glycated hemoglobin levels. Mario Bonadio[2] found a significant association between bacteriuria and glycated hemoglobin levels; but did not find any statistically significant difference in the prevalence of asymptomatic bacteriuria among diabetic and non-diabetic women probably because most of their patients had diabetes that was well controlled.

Escherichia coli was the commonest cause of asymptomatic bacteriuria in women with diabetes in this study. E.coli was responsible for 56 cases among the study group and for 5 in the control group. Makuyana et al[13] found that the commonest bacterial organism isolated in diabetic women was E.coli (26%). Geerlings et al[6] also isolated E.coli as the causative organism in most of their diabetic women in the study group. In the study by Mendoza et al[14], E.coli was the most frequently isolated strain, in 55% of diabetics and 100% of controls. E.coli seems to have unique virulence properties in the urinary tract, including factors promoting adherence to

uroepithelial cells. Geerlings et al[5] in yet another study have found that Type 1 fimbriated E.coli adhere more to diabetic than to control uroepithelial cells. The number of virulence factors in E.coli isolated from the urine of diabetic women with asymptomatic bacteriuria is comparable with the results found in other noncompromised patients with asymptomatic bacteriuria.

The other bacterial organisms isolated from diabetic women with asymptomatic bacteriuria were Klebsiella pneumoniae. Coagulase negative Staphylococci and Proteus mirabilis. Studies by Makuyana et al[13] and Mendoza et al[14] have shown similar results regarding bacterial isolates. Majority of the isolates were susceptible to conventional antibiotics. Certain isolates exhibited some bacterial resistance to the antimicrobials.

No consensus exists regarding the treatment of asymptomatic bacteriuria in diabetic patients. Many experts recommend treating asymptomatic bacteriuria in diabetic patients because of the frequency and severity of upper urinary tract infections. On the other hand few experts believe that the benefit of treatment of asymptomatic bacteriuria is doubtful. This contrast in the result of a lack of follow-

up studies of diabetic women with untreated asymptomatic bacteriuria; At this time, whether diabetic patients with asymptomatic bacteriuria should be treated is not known because whether treatment of asymptomatic bacteriuria prevents the development of symptomatic urinary tract infection or a decline in renal function is not clear. Long-term follow-up studies' will show whether asymptomatic bacteriuria becomes symptomatic and affects renal function in diabetic patients and whether treatment of asymptomatic bacteriuria is warranted.

CONCLUSIONS

In conclusion, this study has shown that the prevalence of asymptomatic bacteriuria is higher in diabetic women than in women without diabetes. The prevalence of asymptomatic bacteriuria in diabetic women was 34% and among non-diabetic women was 8%.

The study has also shown that age is an important risk factor for diabetic women. The age among bacteriuric women was 53.8 ± 12.0 years and among non-bacteriuric women was 51.2 ± 10.6 years ($P < 0.05$).

In the present study, women with longer duration of diabetes had a greater risk of asymptomatic bacteriuria. The duration of diabetes among bacteriuric and non-bacteriuric women was 9.1 ± 6.4 and 7.1 ± 5.7 years respectively.

Poor metabolic control of diabetes was associated with a significant risk of asymptomatic bacteriuria in women with diabetes. Statistically significant elevation of both fasting and postprandial

plasma glucose values was found in bacteriuric diabetic women (Fasting 156.9 + 45.0 and Postprandial 242.8 ± 53.1 mg / dl) when compared with non-bacteriuric diabetic women (Fasting 111.5 ± 34.7 and Postprandial 192. J ± 46.6 mg/dl).

Leucocyturia was found in 82% of positive urine culture (56 out of 68) in the study group comprising of diabetic women and in 83% of positive urine culture (5 out of 6) in the control group comprising of non-diabetic women.

In this study, Escherichia coli was the commonest cause of asymptomatic bacteriuria in diabetic women. Among the 68 bacteriuric women, 56 isolates were E.coli, 6 were Klebsiella pneumoniae , 4 were Coagulase negative Staphylococci and 2 were Proteus mirabilis. Majority of the isolates were susceptible to conventional antibiotics. Certain isolates exhibited some bacterial resistance to the antimicrobials.

SUMMARY

Urinary tract infection is a major disease burden for many patients with diabetes. Asymptomatic bacteriuria is several-fold more common among women with diabetes. The risk of asymptomatic bacteriuria increases with increasing age. The longer the duration of diabetes mellitus, greater is the risk of asymptomatic bacteriuria among diabetic women. A significant impairment of metabolic control of diabetes increases the risk of developing asymptomatic bacteriuria. Escherichia coli is the most common causative organism of asymptomatic bacteriuria in diabetic women. I.ong-term follow up studies will show whether asymptomatic bacteriuria becomes symptomatic and affects renal function in diabetic patients.

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PROFORMA

Name : Age : Sex :

Place : Occupation :

Hospital No. :

Presenting Complaints :

Past History:

Diabetes :

Duration :

Hypertension :

Symptoms of Urinary Tract Infection

Bladder Catheterization During the Past 2 Months

Instrumentation of Urogenital Tract During Past 2 months

Pregnancy

Use of Antimicrobial Drugs During Past 14 days

Recent Hospitalization or Surgery Past 4 Months

Gynaecological Infections

- H/o Vaginal Discharge
- H/o Gental Ulcer
- H/o Pruritus Vulva

Personal History

- Smoking
- Alcohol

Menstrual History

- LMP :
- Period:

General Examination

- Pulse
- Blood Pressure
- Temperature
- Height
- Weight
- BMI

CVS

RS

Abd

CNS

Investigations

- Fasting Plasma Glucose:
- Post Prandial Plasma Glucose:

Urine - Leucocyturia :

Urine - Culture & Sensitivity:

Organism

Anti - Microbial Susceptibility

Ampi	GM	Cotri	Doxy	Cefo	Ceftr	Cipro	Nor	Nali	Amik

ABBREVIATIONS

BMI : Body Mass Index

Sys-BP : Systolic Blood Pressure

Dia-BP : Diastolic Blood Pressure

FPG : Fasting Plasma Glucose

PPPG : Post Prandial Plasma Glucose

ASB : Asymptomatic Bacteriuria

DM : Diabetes mellitus

Dur : Duration

NEJM : New England Journal of Medicine

NIDDM : Non-Insulin dependent Diabetes mellitus